AMERICAN JOURNAL OF BIOMEDICINE AND PHARMACY
https://biojournals.us/index.php/AJBP
ISSN: 2997-7177

Article

The Effect of Fasting on Biochemical Balance in the Human Body: A Review

Jamal A. ALJabbar Attawi*[1]

Gilgamesh University, Baghdad, Iraq
*Correspondence: jamal.a.attawi@gu.edu.iq

Citation: Attawi J. A. A. The Effect of Fasting on Biochemical Balance in the Human Body: A Review. American Journal of Biomedicine and Pharmacy 2025, 2(11), 63-75.

Received: 10th Oct 2025
Revised: 25th Oct 2025
Accepted: 06th Nov 2025
Published: 16th Nov 2025

Abstract: The topic of this review focus on the biochemical effects of fasting, with particular emphasis on metabolic responses that mobilize stored nutrients and preserve homeostasis in times of dearth. Primary adaptive responses include switching to fatty acid and amino acid metabolism (from glucose), the hormonally mediated ability to access additional energy for the brain, and stimulating cellular pathways that support improved energy utilization, stress resistance, and metabolic fitness. Although today's sleep and feeding schedules can be erratic, when people are at least occasionally active then something might restore that metabolic balance. Prolonged fasting might combat the effects of modern life. Studies still delineate fasting's effects, which depend on techniques and inter-individual differences, suggesting the requirement for more research.

Keywords: Biochemical Balance, Liver, Fasting, Metabolism, Insulin

Introduction

Fasting is a willingness to refrain from intake of the body's nourishment for some time, and it has been known since ancient times, practically since humans began to culturally live as people. 1Such remarkable practice correlates with multifarious health benefits, such as longer lifespan and lower incidence of chronic diseases, and becomes an interesting topic in the field [1].

Fasting triggers a series of physiological transformations, particularly involving metabolic alterations aimed at energy conservation and optimal cellular functionality, thus reflecting the body's extraordinary capacity for adaptation. Grasping how these metabolic shifts affect biochemical equilibrium is vital, as this understanding can guide the formulation and endorsement of fasting protocols intended for health maintenance and/or restoration [2].

The goal of this thorough analysis is to bring together all the research that has been done on the biochemical effects of fasting on the human body. The goal is to explain the complex processes that make fasting good for your health. Numerous academic papers and research investigations have thoroughly described the physiological underpinnings of fasting [2].

Fasting usually causes a big change in metabolism from breaking down glucose to breaking down fatty acids. This is followed by a drop in glucose and insulin levels in the blood [3]. This drop in

circulating insulin levels catalyzes lipolysis, a process that leads to an increase in plasma free fatty acids and the creation of ketone bodies as alternative energy sources [4].

At the same time, there is a notable decrease in the amount of amino acids in the blood, mostly due to a decrease in protein intake, which sets off catabolic processes that break down intracellular proteins [5]. Furthermore, oxidative stress and inflammation have been linked to fasting; however, the specifics of these fasting-induced alterations and their allegedly tissue-specific features are still unclear and require more research to fully unravel the complexities. The Scientific Underpinnings of Fasting [6].

Physiological Foundations of Fasting

Deliberately avoiding food for a predetermined amount of time is known as fasting. This phenomenon triggers complex physiological responses in the body, most notably a metabolic shift from the use of glucose to the oxidation of fat for energy production. Hormonal concentrations that regulate different biochemical processes are impacted by such a transition. Furthermore, a variety of cellular and molecular pathways essential to the fields of biochemistry, physiology, and behavioral regulation are stimulated by fasting [7]. A thorough grasp of the biochemical foundations of fasting practices is established by the scientific literature's strong documentation of the physiological reactions elicited by fasting [4].

The liver, an organ central to metabolism and the regulation of glucose and lipoprotein homeostasis, is profoundly affected by fasting. In a mammal that is fasting for an extended period, blood glucose levels drop due to decreased intake and increased consumption of glucose stemming from glycerol and gluconeogenesis [1]. With the destruction of glycogen stores in the liver, triglycerides become a major energy source, and lipolysis becomes the primary catabolic pathway. Fatty acids broken down by lipolysis are transported to and utilized in the liver, which converts free fatty acids into acyl-CoA and subsequently into acetyl-CoA. Acetyl-CoA formed during fasting is not utilized for cholesterol synthesis [8].

A. Metabolic Shift During Fasting

Fasting induces a general metabolic shift. The use of carbohydrates for energy is sharply reduced. Free fatty acid concentration rises in the plasma, and fatty acids are increasingly converted to ketone bodies. Ketone bodies invade the brain, replacing glucose as an energy source. Substantial amounts of free amino acids are released from muscle and other protein pools, and ammonia is produced from proteins or amino acids at a high rate [2].

Fasting causes the breakdown of triglycerides into free fatty acids and glycerin. Glycerin can be changed to glucose via gluconeogenesis, and free fatty acids can be transformed into ketone bodies and utilized by other organs. After prolonged fasting, energy sources shift from glucose to insulin exacerbation and, eventually, to triglycerides and proteins [6].

B. Hormonal Regulation

Hormonal levels are significantly altered during fasting, which in turn affects important bodily physiological functions. Notably, growth hormone levels rise in tandem with a drop in insulin levels and an increase in glucagon levels [9]. These hormones have a central role in the metabolic use of energy and nutrients. In short periods of fasting, changes in hormone levels affect energy metabolism in various ways [10]. In particular, research indicates that fasting elevates levels of steroid hormone precursors while simultaneously reducing levels of certain steroids, such as androgens, in healthy young women. This response indicates that fasting may improve the body's capacity to regulate energy substrates, thereby preserving homeostasis and facilitating survival in the absence of food

consumption. A thorough understanding of these hormonal fluctuations is essential for elucidating the impact of fasting on health and physiological function [11].

C. Cellular and Molecular Pathways

Fasting causes big changes in metabolism because the body goes from being fed to being fasted. This metabolic change can be broken down into three parts: (1) the substrate mobilization phase, which lasts about six hours; (2) the gluconeogenesis phase, which lasts about three days; and (3) the ketogenic phase, which can last for weeks [12]. The time of the last meal is called "hour zero," and the blood glucose level is about 5 mM at that time. When the body goes into a fasted state, the levels of glucose and insulin in the blood go down, and the levels of the catecholamines epinephrine and norepinephrine go up.Insulin concentrations reach a nadir between 12 and 24 hours into the fasting process, while glucose levels decrease to approximately 4 mM [13].

Adipose triglycerides undergo hydrolysis to free fatty acids (FFAs) and glycerol, while hepatic glycogen degradation begins to support gluconeogenesis [13]. By hour 6, liver glucose production becomes significant enough to compensate for whole-body utilization, and urea and lactate production rates adapt to the new metabolic state. In roughly three days, there is a reduction in systemic glucose utilization to 50% of the value observed during the fed state. The turnover rate of the glucose pool changes to once every 24 hours, as opposed to occurring every few hours, while the glycogen content within the liver declines to less than 2% of the organ's total mass [11].

These metabolic modifications are associated with a systematic array of hormonal transformations. The main hormone produced in the gut that contributes to the biological rhythms brought on by food consumption is glucose-dependent insulinotropic peptide. Other gut-derived hormones, such as glucagon-like peptide-1, peptide YY, and somatostatin, are elevated during fasting. Furthermore, because gluconeogenic substrates like alanine, lactate, and glycerol are preferentially diverted towards intestinal metabolism rather than hepatic metabolism during fasting, there are notable changes in growth hormone, glucagon, and insulin-like growth factor 1 [14].

D. Biochemical Effect Induced by Fasting

As the process of fasting proceeds, significant biochemical changes occur in the human body. More particularly, there is a decrease in glucose and insulin levels, as well as an increase in lipids and ketone bodies, which indicate that the organism is shifting toward metabolic adaptation due to nutritional deprivation. Plasma amino acid concentration is also altered, with a significant decrease in essential amino acids, which are necessary for various physiological functions [7]. However, nitrogen balance improves, as marked by decreased urea levels, reflecting the body's efficiency in better utilization of protein. Moreover, the redox balance shifts towards a more reduced state that might trigger an evolutionarily conserved cytoprotective mechanism aimed at maintaining cellular homeostasis. The biomarkers of inflammation generally show a trend toward reduction, with TNF-α and CRP emerging as key signaling molecules during this process. Moreover, fasting promotes the up-regulation of immune cells and thus defends the body against pathogenic organisms, thereby helping maintain general health [15].

E. Dynamics of Glucose and Insulin During Fasting

Fasting induces a chain of endocrine and metabolic responses to altered fuel availability. The availability of circulating glucose is crucial to cellular metabolism and homeostasis. Glucose-enriched interstitial fluids act as extracellular mediators in this metabolic pathway. As the duration of fasting increases, there is a metabolic transition from utilizing glucose to incorporating fatty acids and ketone bodies [11].

The principal effect of glucose and insulin is the regulation of metabolic pathways linked to the cells' bioenergetics by controlling the glycolytic and pentose-phosphate pathways and modulating the

cellular redox state. The liver and the brain provide a dual control mechanism to adjust glucose levels within the physiological range; glucose plays a major role in several cell types, such as pancreatic β cells, where glucose mediates insulin secretion in an attending way [16]. The necessity of an additional energy substrate arises when the fasting period exceeds 8 hours. The physiological reaction to fasting solely consisted of a decrease in the daily glycaemia without any other metabolic imbalance; the human body is capable of alternating between day and night fasting cycles [17].

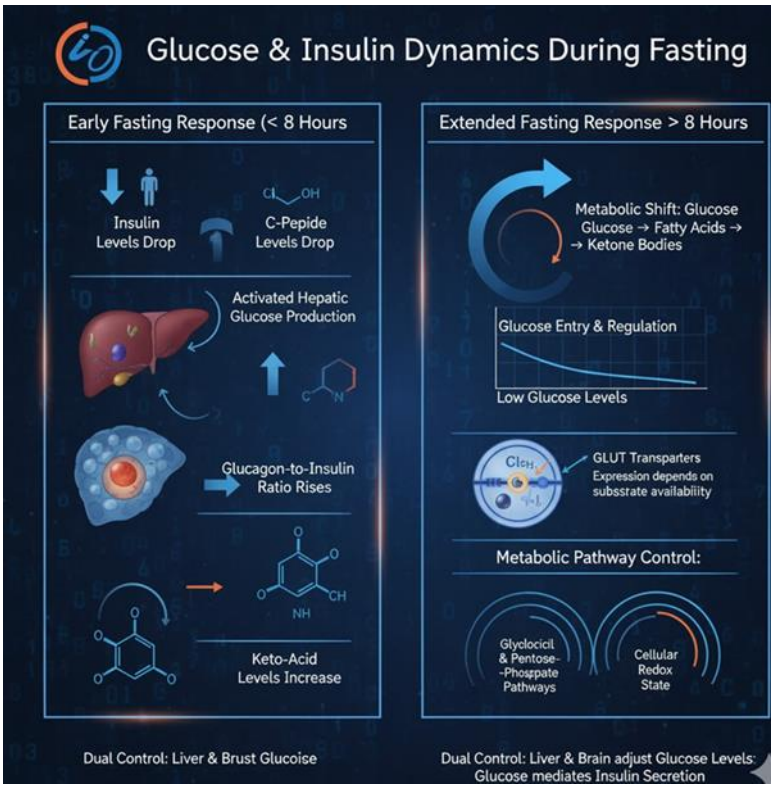

Figure 1. Glucose and Insulin Dynamics during Fasting.

F. Lipid Metabolism and Ketogenesis

The metabolism of lipids is closely linked to energy expenditure, oxidative stress, and inflammation. After a meal, triglycerides are stored in adipose tissue. During fasting, adipose tissue begins to release free fatty acids, which are taken up by the liver [18]. In the liver, fatty acids undergo beta-oxidation, generating acetyl-CoA. In a prolonged fasting state, as energy reserves deplete, the liver converts excess acetyl-CoA into ketone bodies (acetoacetate, β-hydroxybutyrate, and acetone), which then enter the bloodstream [10]. Blood levels of ketone bodies increase within 8–12 hours of fasting and reach 2–5 mM after 24 hours. Ketone bodies serve as an alternative energy source and play a critical role in maintaining energy homeostasis [17].

Fasting induces a complex network of biochemical processes, the detailed mapping of which is crucial to elucidating the systemic physiological responses to fasting and establishing the mechanisms through which fasting exerts its beneficial effects on health [17].

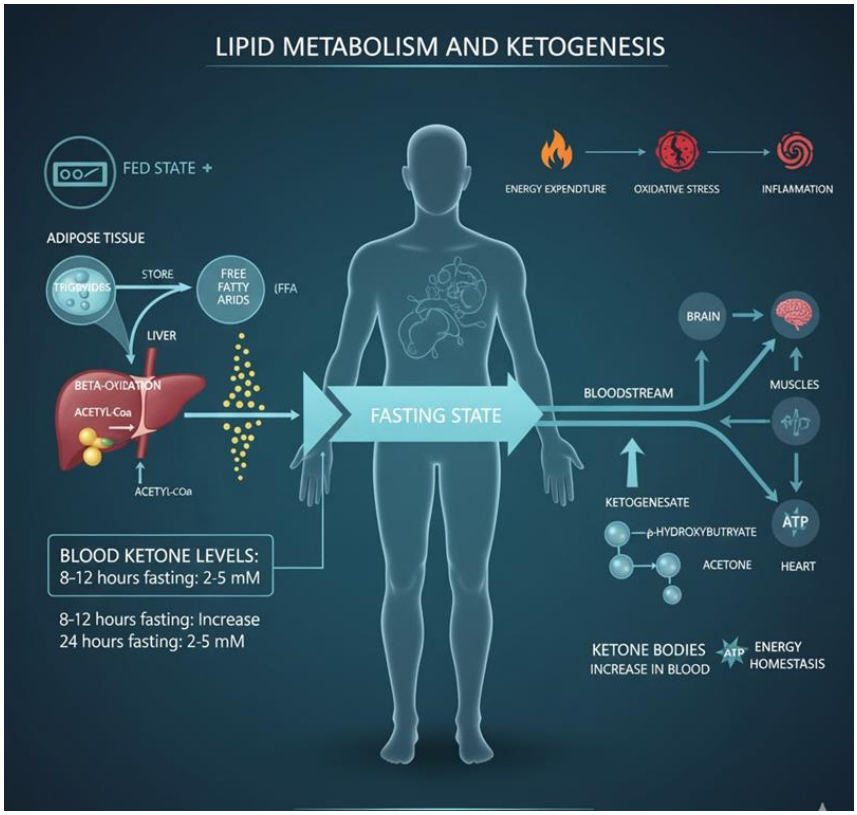

Figure 2. Lipid metabolism and ketogenesis.

G. Amino Acids and Protein Turnover

When fasting is imposed experimentally for up to 3 days, rates of protein synthesis gradually decline so that by 72 hours these rates are approximately 15 to 20 per cent less than those prior to fasting [19].

In a normal man, both fasting and refeeding influence the rates of protein breakdown and synthesis, but the adaptive responses occur at different times. After an overnight fast synthesis of plasma proteins decreases [20]. Prior to feeding, there is a decrease in the concentration of metabolic-stimulating agents, with a concomitant fall in the ratio of free fatty acid to triglyceride. Feed from the minor sources of protein in the diet shifts rapidly to a major exogenously derived input when at least 250 kcal are ingested, and within 4 h, the rate of plasma protein synthesis, maintained at a nearly constant level, begins to be raised. Limited reports suggest that prolonged fasting affects muscle protein. Following 6 days of fasting, muscle metabolism remains at the prefasting level, but liver protein synthesis is decreased [21].

Different forms of fasting influence plasma free amino acids differently. After 4 h of fasting, circulating free amino acids are significantly altered. A heavier and larger adaptive range occurs in the plasma free amino acid pool during protein restriction compared to the simultaneous elevation of this circulating pool observed during fasting. The free amino acids from protein breakdown during prolonged fasting appear gradually to be diverted more preferentially to specific organ systems such as the liver and kidney [22].

H. Oxidative Stress and Redox Homeostasis

The human body relies on a variety of nutrient sensors to adapt to physiological extremes. Caloric restriction, fasting, and even long periods of prolonged hunger elicit adaptations that promote longevity and lifespan extension [23]. During the first hour to ten hours of fasting, the body begins to modify its redox homeostasis and glucose metabolism, which then leads to metabolic shifts as it adapts

to the absence of exogenous nutrients [24]. The organism employs an array of oxidation-reduction (redox) circuitry, stored nutrients, metabolic reactions involving the mitochondria, and orally consumed fats in order to maintain the continual supply of energy that is imperative for sustaining life. Under fasting conditions, biomolecules that accumulate in the bloodstream such as ketone, lipid, urea, and level changes of glucose tend to reach stable levels that match nutrient intake from food [25].

In a study investigating fasting-induced oxidative stress, the plasma parameters of individuals who fasted during Ramadan were characterised, identifying malondialdehyde (MDA), total antioxidant capacity (TAC), and other biomarkers that play important roles in the defence mechanism, cellular signalling, and regulating cellular redox homeostasis. Results correlated with increased plasma TAC in the fasting group. Fasting also reduced serum levels of some of the main parameters that are upregulated by the ingestion of extra calories, including glucose, triglycerides, and low-density lipoprotein cholesterol [26].

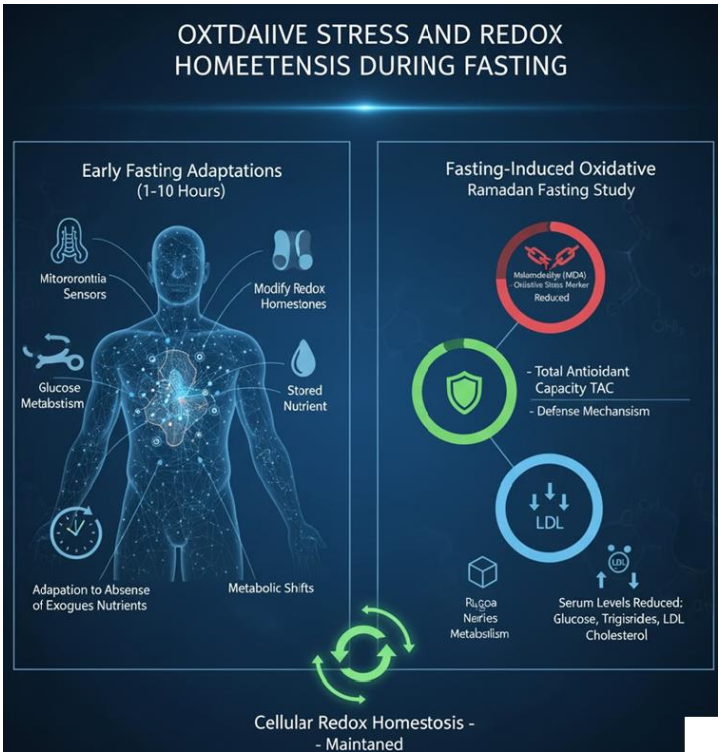

Figure 3. Oxidation stress and redox homeostasis during fasting.

I. Inflammatory Markers and Immune Function

Fasting affects immune function and levels of inflammatory mediators through diverse mechanisms. A recurrent circadian fasting regimen reduced plasma tumor necrosis factor alpha (TNF-α) but elevated interleukin (IL)-6 concentrations [27]. During fasting, older adults remained resistant to TNF-α elevations, highlighting an age-related adaptation. Inflammatory mediators associated with the senescence-associated secretory phenotype (SASP) persisted despite fasting, paralleling IL-6 patterns. Overall, chemokines related to T-helper cells, B lymphocytes, and monocytes exhibited diminished expression. Interleukin-1β signaling became counterbalanced by circulating IL-1 receptor antagonist (IL-1RA), which binds competitively to the IL-1 receptor [28].

Fasting Regimens and Biochemical Outcomes

Fasting regimens can produce distinct alterations in human biochemistry. Intermittent fasting (IF) schemes allow eat–stop–eat dietary cycles during a 24-h day, while time-restricted eating (TRE)

schedules pair moderate time-restricted eating windows, typically between six to nine hours, with an extended fasting period within the day [29]. Prolonged fasting (PF) regimens often consist of multi-day fasting without solid food but with water intake, and can also include either moderate caloric intake with single or multi-day extended fasting. Caloric restriction (CR) however, remains distinct both chemically and biochemically from fasting. Controlled studies assessing fasting regimens report that IF and TRE did not influence body weight control or metabolic indices in overweight and obese adults [30]. Biochemical parameters directly associated with glucose–insulin metabolism including fasting glucose, postprandial glucose, and 2-h OGTT did not significantly differ between fasting and control. Important CrP markers were substantially lower in those fasting compared to those on a traditional diet of restricted caloric intake. Also, PAI-1, ADPN and TRI were lower in one group of subjects after a 2-week fasting period. These metabolically relevant changes specifically observed during PF, in triglycerides for example, occur in parallel with much broader alterations in the hepatic biochemistry and physiology [31]. Studies also show that multi-day fasting induces a change in energy supplier, lessening dependence on fatty fuel reserves and changing the profile of plasma free acids, amino acids, and lipids. Metabolic flexibility or the capacity to utilize and switch from various energy sources is normally reduced in chronic metabolic disease and tightly linked to the risk gradient of obesiogenic disease outcomes [30], [31].

A. Intermittent Fasting

Intermittent fasting (IF) has gained popularity as an alternate approach to weight reduction and management of lifestyle-associated metabolic disorders. In addition to its widespread use in weight and health management, IF is also being studied for its effects on human athletic performance. IF comprises time-restricted feeding such that eating is limited to a continuous window of 4 to 12 hours daily and the remainder of the day is reserved for fasting [32].

The fasting duration permitted by IF facilitates a metabolic switch to fat burning for energy, where the preferred metabolic fuel source transitions from glucose and carbohydrates to fatty acids and ketone bodies. IF is commonly perceived to confer benefits on weight loss, maintaining fat loss, and metabolic health through enhanced fat oxidation, improved lipid profile, lipid metabolism, and a favorable hormonal milieu. Nonetheless, evidence suggests that the short-term effects of fasting regimens on biochemical parameters are preserved (i.e., during 8 hours of fasting before breakfast) [33].

B. Time-Restricted Eating

Time-restricted eating is a time-based eating regimen regulated through restricting food intake to specific time frames. Time-restricted eating is reported to improve nutritional health, including body composition, cardiometabolic risk factors and metabolic diseases such as diabetes mellitus [34]. Because time-restricted eating does not prohibit or promote a specific food intake, research focuses on the effect of time-restricted eating on glucose and insulin metabolism. Time-restricted eating appears to have a beneficial influence on insulin dynamics and the regulation of glucose homeostasis, and its diffusion to the general public is increasing [29].

Fasting regimens such as intermittent fasting (sometimes called alternate-day fasting) include fasting on alternate days or fasting 1 or 2 days each week without restriction on food intake during non-fasting days. Intermittent fasting exerts favourable effects on several metabolic diseases and conditions. Intermittent fasting cannot be extrapolated to time-restricted eating because of the difference in fasting duration. Prolonged fasting (the duration of food withdrawal must exceed 48 h before considering a fast prolonged), on the other hand, cannot be directly compared with time-restricted fasting because the biochemical alterations differ significantly [34].

Food-taboo culture such as Ramadan fasting or Yom Kipur is common in many countries. Food-restriction during early daytime is considered a continuous physiological fasting state that ensures

continuity from the previous night's rest. Continuous food restriction up to 12 h or longer between meals may become fasting-research centres of interest. Additional scientific studies on the influence of food-control patterns relative to duration of large calorific meals on metabolic improvement should be carried out to broaden the understanding [35].

C. Caloric Restriction Versus Fasting

Caloric restriction (CR) implies the long-term reduction of caloric intake without malnutrition, while fasting represents a temporary interruption of caloric intake. CR was shown to extend lifespan and delay aging markers across different species, but its efficacy in humans remains uncertain. Studies indicate that CR reduces several biomarkers of aging in humans without causing negative psychological effects. Long-term CR lowers insulin and insulin-like growth factor-1 (IGF-1), enhancing the insulin signaling pathway, which increases the expression of sirtuin proteins, ultimately leading to life-span extension [36]. Fasting reduces growth hormone (GH) and IGF-1 more than CR does. In Ammonia-Dependent Regulation of Cross-Talk Between the Urea and Polyamine Cycles and Relevance to Aging, Ganesan suggests that fasting inhibits the mechanistic target of rapamycin (mTOR) in a manner that differs from CR. Several studies indicate that fasting, unlike CR, upregulates endogenous metabolic cofactors, including nicotinamide adenine dinucleotide (NAD+), coenzyme A (CoA), and 5′-adenosine monophosphate (AMP), which increase metabolic plasticity and counter cardiac aging through restoring mitochondrial function and enhancing stress-resistance life-extending pathways [37].

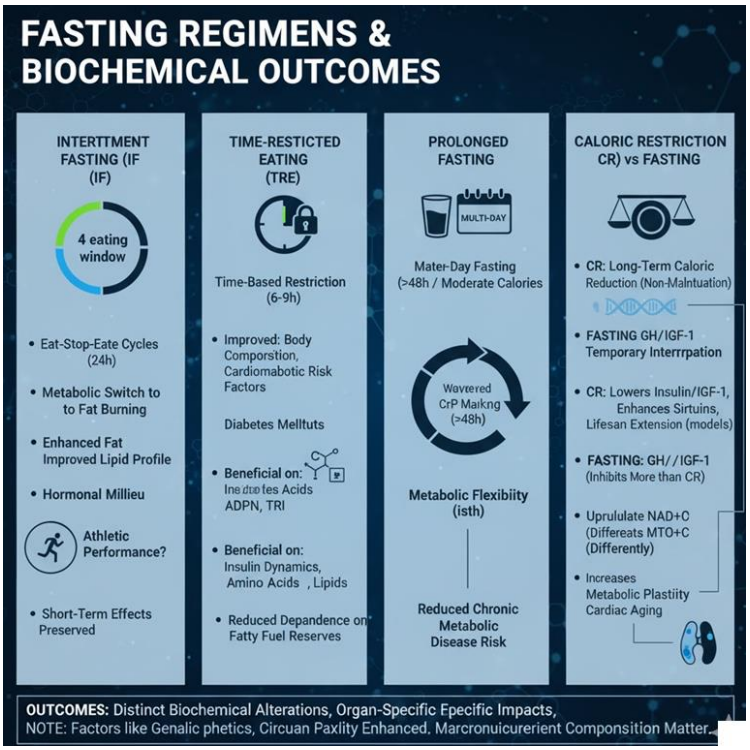

Figure 4. Fasting regimens and biochemical outcomes.

Organ-Specific Biochemical Effects

Fasting exerts multiple organ-specific impacts because it increases substrate availability, notably fatty acids and ketone bodies and alters substrate preference, influencing key metabolic and signaling pathways across various tissues [38]. In the liver, fasting lowers glucose levels and insulin secretion, stimulating lipolysis and hepatic fatty acid uptake; fatty acids are subsequently oxidized or converted into ketone bodies, which circulate to other organs. Fasting also elevates hepatic phosphatidylcholine, a structural component of membranes that buffers excess free cholesterol, while pool sizes of neutral

amino acids—sources of carbon, nitrogen, and reducing factors for function and metabolism—decline; leptin and total tricarboxylic acid cycle intermediates are similarly diminished [39].

Muscle and adipose tissue utilize similar energy sources. During the first 24 hours of fasting, muscle glucose oxidation decreases, triglycerides stored in adipose tissue are cleaved into free fatty acids and glycerol, and hepatic enzymes for fatty acid synthesis become inactive 1. The activity of the pentose phosphate pathway and the levels of cytosolic free ribonucleotides, key determinants of protein turnover, remain unchanged, in contrast to the decrease in protein synthesis rate and the increase in protein degradation rate. Similarly, in the brain, fasting extends the time before energy-consuming processes adapt to the limited energy supply. Along with a reduction in insulin levels, brain levels of phosphatidylethanolamine, arachidonic acid, sphingomyelin, free cholesterol, leptin, acetyl-CoA, and adrenaline drop; glucose oxidation remains limited even after several days of fasting [38].

A. Hepatic Metabolism

Fasting induces profound biochemical alterations in the human body, prominently in the liver. Various fasting regimens produce distinct metabolic adaptations. Responses are also shapeed by numerous factors, including the duration of fasting, macronutrient composition of meals before fasting, environmental temperature, circadian phases, and genetic background [38]. Further exploration of these adaptations, and hepatic metabolism in general, will provide a more detailed and unbiased review of the metabolic consequences of fasting than is possible in this review. The liver remains a critical player in overall metabolism during fasting. Fasting triggers a chain of events in hepatic energy metabolism, including increased fatty acid use, increased glucose production, and enhanced production of fibroblast growth factor 21 (FGF21) [37]. Prolonged fasting triggers unique metabolic pathways. After 18 hours of fasting, mice exhibit a robust increase in plasma non-esterified fatty acids (NEFAs). Increased uptake of NEFAs elicits energetic stress and degradation of the intermediate metabolite acyl-CoA, leading to metabolic reprogramming characterized by elevation of plasma β-hydroxybutyrate levels 1 [40].

B. Muscle and Adipose Tissue

The most profound metabolic adaptation to fasting occurs in skeletal muscle and adipose tissue. In humans, the maximal decline in glucose concentrations is observed after 10 to 12 hours of fasting, with the greatest increase in free fatty acids occurring within 4 to 6 hours and the alternate elevation of β-hydroxybutyrate beginning after 12 hours 15.9 Gene programs controlling lipid and glucose metabolism in isolated human skeletal muscle cells show that fasting subjects have elevated genes engaged in lipolysis and use of fatty acids for energy, while the reverse is true in subjects maintained on a high-carbohydrate diet. After 2 days of fasting, there is a further increase in fasting genes, and the program appears to be conserved [39].

On the other hand, both de novo lipogenesis in the liver and adipose tissue and cholesterogenesis in adipose tissue, regulated by the insulin-mTORC1-SREBP axis, drop with the onset of fasting, while lipophagy increases. Changes in adipose tissue gene expression after prolonged fasting reflect activation of the PPARγ–PGC1α–PRDM16–UCP1 axis. Therefore, fasting induces complex, tissue-specific alterations on the transcriptional level across the human body, involving diverse aspects of metabolism and leading to widespread metabolic reprograming [41].

C. Neurobiological Biochemistry

Fasting initiates a series of neurobiochemical changes, thereby affecting psychological well-being. Several studies explore the relationship between fasting and mental health. First, intermittent fasting has been associated with improved cognitive function and learning [42]. Neurobiological signaling through insulin, brain-derived neurotrophic factor (BDNF), neuropeptide Y, sirtuins, and silent information regulator type 1 (SIRT1) are implicated [42]. Prolonged fasting reduces the levels of

serum norepinephrine, epinephrine, and dopamine, whereas the levels of glucagon, growth hormone, and serotonin increase. After 24 hours of fasting, the expression of neuropeptide Y gene in the hypothalamus is enhanced. These fasting-induced biochemical changes may largely contribute to altered psychological health [43].

The historical use of fasting stresses its significance in this respect. Anthropological studies indicate that early Homo sapiens experienced starvation from both natural disasters (e.g., drought or wildfire) and the seasonal availability of food. During periods of enforced fasting arising from these circumstances, early humans engaged in voluntary fasting [44]. The worldwide practices of fasting for religious purposes also sustain the notion of fasting as a natural but largely neglected human activity. The recent upsurge of scientific research into various aspects of fasting and food deprivation has brought attentions to its health benefits and has consolidated the idea of fasting as a therapeuetic intervention against various disorders [15].

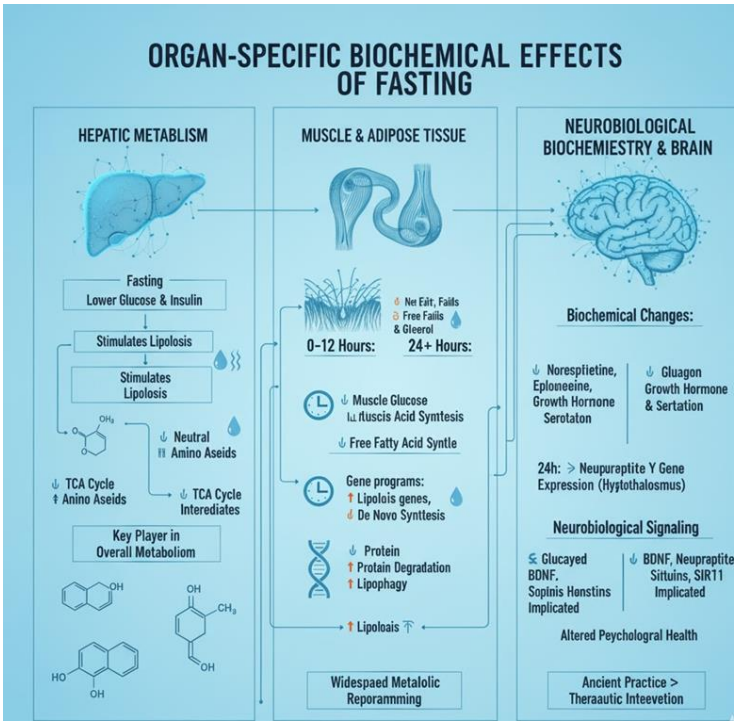

Figure 5. Organ-specific biochemical effect of fasting.

Conclusion

Fasting is a natural biological process that has a substantial impact on the biochemical equilibrium in the human body and sustains health. It is a monitored abstention from food, beverages containing calories, or any consumables during the day or for multi-day periods. Various fasting paradigms exist that differ in terms of duration, energy deprivation, or frequency, and the specific biochemical and physiological responses during fasting depend on its characteristics. Such fasting regimens as intermittent fasting (IF), time-restricted eating (TRE), prolonged fasting (PF), and caloric restriction (CR) evoke different biochemical alterations at varying intensities and may extend longevity and healthspan in organisms ranging from yeasts and worms to flies and rodents. Studies indicate that fasting alters simultaneously the biochemical dynamics of insulin and glucose, lipids and ketone bodies, amino acids and proteins, oxidative stress, redox balance, inflammatory processes, and many other markers. Due to these conditions, fasting and its regimen-associated particular effects are under investigation regarding their pharmacodynamic, therapeutic, and health-related uses in humans.

REFERENCES

[1] P. Horn and F. Tacke, "Metabolic reprogramming in liver fibrosis," *Cell Metabolism*, vol. 36, no. 7, pp. 1439–1455, 2024.

[2] S. Kersten, "The impact of fasting on adipose tissue metabolism," *Biochim. Biophys. Acta Mol. Cell Biol. Lipids*, vol. 1868, no. 3, p. 159262, 2023.

[3] H. Kondoh, T. Teruya, and M. Yanagida, "Metabolomics of human fasting: new insights about old questions," *Open Biol.*, vol. 10, no. 9, p. 200176, 2020.

[4] D. A. Koppold *et al.*, "International consensus on fasting terminology," *Cell Metabolism*, vol. 36, no. 8, pp. 1779–1794.e4, 2024.

[5] B. P. Magyar *et al.*, "Short-term fasting attenuates overall steroid hormone biosynthesis in healthy young women," *J. Endocr. Soc.*, vol. 6, no. 7, p. bvac075, 2022.

[6] P. M. Ruppert and S. Kersten, "Mechanisms of hepatic fatty acid oxidation and ketogenesis during fasting," *Trends Endocrinol. Metab.*, vol. 35, no. 2, pp. 107–124, 2024.

[7] Y. Wang and R. Wu, "The effect of fasting on human metabolism and psychological health," *Disease Markers*, vol. 2022, no. 1, p. 5653739, 2022.

[8] Y. Wang, "Triglycerides, glucose metabolism, and type 2 diabetes," *Int. J. Mol. Sci.*, vol. 26, no. 20, p. 9910, 2025.

[9] A. Attinà *et al.*, "Fasting: How to guide," *Nutrients*, vol. 13, no. 5, p. 1570, 2021.

[10] S.-L. Li *et al.*, "Restoration of HMGCS2-mediated ketogenesis alleviates tacrolimus-induced hepatic lipid metabolism disorder," *Acta Pharmacol. Sin.*, vol. 45, no. 9, pp. 1898–1911, 2024.

[11] A. F. Vieira *et al.*, "Impact of overnight fasted state versus fed state on adaptations to resistance training: A randomized clinical trial," *Int. J. Sport Nutr. Exerc. Metab.*, vol. 35, no. 4, pp. 291–302, 2025.

[12] N. Vo, Q. Zhang, and H.-K. Sung, "From fasting to fat reshaping: exploring the molecular pathways of intermittent fasting-induced adipose tissue remodeling," *J. Pharm. Pharm. Sci.*, vol. 27, p. 13062, 2024.

[13] M. P. Mattson, "The cyclic metabolic switching theory of intermittent fasting," *Nat. Metab.*, pp. 1–14, 2025.

[14] C. Reytor-González *et al.*, "From meal to malfunction: exploring molecular pathways, biomarkers and interventions in postprandial cardiometabolic health," *Front. Cardiovasc. Med.*, vol. 12, p. 1655889, 2025.

[15] S. Min *et al.*, "The association between regular physical exercise, sleep patterns, fasting, and autophagy for healthy longevity and well-being: a narrative review," *Front. Psychol.*, vol. 12, p. 803421, 2021.

[16] S. Y. Park, J.-F. Gautier, and S. Chon, "Assessment of insulin secretion and insulin resistance in human," *Diabetes Metab. J.*, vol. 45, no. 5, pp. 641–654, 2021.

[17] D. Tang *et al.*, "Fasting: from physiology to pathology," *Adv. Sci.*, vol. 10, no. 9, p. 2204487, 2023.

[18] R. Suarez *et al.*, "Very low calorie ketogenic diet: What effects on lipid metabolism?" *Curr. Nutr. Rep.*, vol. 13, no. 3, pp. 516–526, 2024.

[19] N. Winter, M. Novatchkova, and A. Bachmair, "Cellular control of protein turnover via the modification of the amino terminus," *Int. J. Mol. Sci.*, vol. 22, no. 7, p. 3545, 2021.

[20] K. J. Paulussen *et al.*, "Anabolic resistance of muscle protein turnover comes in various shapes and sizes," *Front. Nutr.*, vol. 8, p. 615849, 2021.

[21] C. Laurens *et al.*, "Is muscle and protein loss relevant in long-term fasting in healthy men? A prospective trial on physiological adaptations," *J. Cachexia Sarcopenia Muscle*, vol. 12, no. 6, pp. 1690–1703, 2021.

[22] R. Karimi *et al*., "Adaptive effects of endocrine hormones on metabolism of macronutrients during fasting and starvation: A scoping review," *Metabolites*, vol. 14, no. 6, p. 336, 2024.

[23] M. Barteková *et al*., "Natural and synthetic antioxidants targeting cardiac oxidative stress and redox signaling in cardiometabolic diseases," *Free Radic. Biol. Med.*, vol. 169, pp. 446–477, 2021.

[24] K. Le Gal, E. E. Schmidt, and V. I. Sayin, "Cellular redox homeostasis," *Antioxidants*, vol. 10, no. 9, p. 1377, 2021.

[25] O. H. Hewitt and S. M. Degnan, "Distribution and diversity of ROS-generating enzymes across the animal kingdom, with a focus on sponges (Porifera)," *BMC Biol.*, vol. 20, no. 1, p. 212, 2022.

[26] M. Brazaitis *et al*., "Six-day fasting causes temporary increases in both antioxidant capacity and oxidative stress in healthy young men: A randomized controlled trial," *Antioxidants*, vol. 14, no. 3, p. 269, 2025.

[27] A. Mulas *et al*., "Effect of intermittent fasting on circulating inflammatory markers in obesity: A review of human trials," *Front. Nutr.*, vol. 10, p. 1146924, 2023.

[28] C. Wilhelm, J. Surendar, and F. Karagiannis, "Enemy or ally? Fasting as an essential regulator of immune responses," *Trends Immunol.*, vol. 42, no. 5, pp. 389–400, 2021.

[29] G. Różański *et al*., "Effect of different types of intermittent fasting on biochemical and anthropometric parameters among patients with metabolic-associated fatty liver disease (MAFLD)—A systematic review," *Nutrients*, vol. 14, no. 1, p. 91, 2021.

[30] Y. A. Mohamed *et al*., "Effect of intermittent fasting on lipid biokinetics in obese and overweight patients with type 2 diabetes mellitus," *Diabetol. Metab. Syndr.*, vol. 16, no. 1, p. 4, 2024.

[31] N. K. Qureshi, N. Akter, and Z. Ahmed, "Follow-up and treatment pattern during Ramadan …" *BIRDEM Med. J.*, vol. 11, no. 1, pp. 30–38, 2021.

[32] U. A. Shah and N. M. Iyengar, "Plant-based and ketogenic diets as diverging paths to address cancer: a review," *JAMA Oncol.*, vol. 8, no. 8, pp. 1201–1208, 2022.

[33] R. Herodek, M. Živković, and A. Catić-Đorđević, "Changes in body composition induced by Orthodox religious fasting on high school students," *Facta Univ. Ser. Teach. Learn. Teach. Educ.*, pp. 037–046, 2024.

[34] M. E. Parrotta *et al*., "Time restricted eating: A valuable alternative to calorie restriction for addressing obesity?" *Curr. Obes. Rep.*, vol. 14, no. 1, p. 17, 2025.

[35] J. Welbourne, R. Heath, and D. Martin, "Daytime only or time restricted feeding in critically ill patients," *J. Intensive Care Soc.* 2025.

[36] S. J. Hofer *et al*., "The ups and downs of caloric restriction and fasting: from molecular effects to clinical application," *EMBO Mol. Med.*, vol. 14, no. 1, p. e14418, 2022.

[37] H. H. Pak *et al*., "Fasting drives the metabolic, molecular and geroprotective effects of a calorie-restricted diet in mice," *Nat. Metab.*, vol. 3, no. 10, pp. 1327–1341, 2021.

[38] Y. Ahmad, D. S. Seo, and Y. Jang, "Metabolic effects of ketogenic diets," *Int. J. Mol. Sci.*, vol. 25, no. 13, p. 7076, 2024.

[39] P. Puchalska and P. A. Crawford, "Metabolic and signaling roles of ketone bodies in health and disease," *Annu. Rev. Nutr.*, vol. 41, pp. 49–77, 2021.

[40] P. M. Ruppert and S. Kersten, "Mechanisms of hepatic fatty acid oxidation and ketogenesis during fasting," *Trends Endocrinol. Metab.*, vol. 35, no. 2, pp. 107–124, 2024.

[41] Y. Fan *et al*., "Uncovering shared and tissue-specific molecular adaptations to intermittent fasting," *bioRxiv*, preprint, 2025.

[42] E. Hosseini *et al*., "Fasting diets: what are the impacts on eating behaviors, sleep, mood, and well-being?" *Front. Nutr.*, vol. 10, p. 1256101, 2024.

[43] K. Stec *et al*., "Effects of fasting on physiological and psychological responses in middle-aged men," *Nutrients*, vol. 15, no. 15, p. 3444, 2023.

[44] J. Gudden, A. Arias Vasquez, and M. Bloemendaal, "The effects of intermittent fasting on brain and cognitive function," *Nutrients*, vol. 13, no. 9, p. 3166, 2021.